REVIEW OF THE WORK DONE AT ICRISAT ON SOIL-BORNE DISEASES OF PIGEONPEA AND CHICKPEA

Y. L. NENE, J. KANNAIYAN, M. P. HAWARE, AND M. V. REDDY

Prepared for the Consultants' Group Discussion on
the Resistance to Soil-borne Diseases of Legumes

(January 8-11, 1979)

ICRISAT

International Crops Research Institute for the Semi-Arid Tropics
1–11–256 Begumpet
Hyderabad 500016 (A.P.) India

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Y.L. Nene, J. Kannaiyan, M.P. Haware, and M.V. Reddy

Work on the pathology of ICRISAT's two pulse crops, pigeonpea (*Cajanus cajan* (L.) Millsp.) and chickpea (*Cicer arietinum* L.), was initiated in September 1974 after one of us (YLN) joined as the Principal Pathologist. According to the requirement of the Institute, a seminar on the proposed plan of work was presented by YLN in November 1974. He stated in his seminar that the objective of the pathology program should be to play an appropriate role (i) in the Crop Improvement Program by providing assistance in breeding disease-resistant material and (ii) in maintaining the gains made in the Crop Improvement Program. Subsequently we planned our research projects and all along we have kept in mind the above two objectives. While the first objective would explain all our work on screening techniques and their application, the second objective would explain our work on relevant aspects of biology and epidemiology of the pathogens concerned. The phrase 'soil-borne diseases' can cover several diseases. We have, however, stuck to more commonly accepted connotation and that should explain our coverage in this review.

PIGEONPEA

I. Wilt

1. Introduction

A very large number of papers on highly varied aspects have

appeared in the literature since the disease was first described from India by Butler in 1906. In 1910 he described in detail pathogenicity experiments and also described the causal fungus to be a new species of *Fusarium, F. udum*. Even though attempts have been made to change the fungus name to *F. oxysporum* f. sp. *udum*, we agree with Booth (1971) and stick to the name *F. udum*. It is fairly easy to distinguish *F. udum* from *F. oxysporum* on the basis of spore morphology. An attempt to identify wilt-resistant lines was initiated as early as 1905 at Poona in India (Butler, 1908, 1910).

2. Occurrence

The disease is widely prevalent in India (Butler 1906). It has consistently been reported to be more serious in central and northern India.

The disease has been reported/observed in Kenya, Tanzania and Uganda in Africa, Thailand and Indonesia in South-East Asia, and Trinidad in the Caribbean. Seriousness of the disease in these countries, however, is doubtful.

3. ICRISAT surveys

There are no two opinions about the seriousness of this disease in India. Several workers have made general statements on the widespread occurrence of the disease and the serious losses that it causes. We have not, however, come across any report of a systematic survey of this disease. In 1975 we started roving surveys in cooperation with agricultural universities in different states in India. To date we have surveyed five states covering over 18,000 km. Stops were made

approximately between 30 to 40 km, except in non-pigeonpea growing areas. The data at each stop were collected using a standard proforma which ensured uniformity in data collection. The results obtained so far are summarized in Table 1.

Table 1. Pigeonpea wilt survey (1975-1977)

State	Distance covered km	Locations	Districts	Average %	Range in farmers' fields %
Andhra Pradesh	4,000	102	19	5.26	0-92
Maharashtra	4,000	82	19	22.61	0-93
Karnataka	2,000	37	14	1.12	0-17
Tamil Nadu	2,100	46	11	1.36	0-65
Madhya Pradesh	6,000	136	40	5.42	0-96

These surveys confirm the presence of the disease in every state surveyed so far, with relatively more in central India. We have yet to conduct surveys in the three major northern states of India.

4. Loss estimation

It was generally presumed that every wilted plant represents total loss. Since we see (i) partial wilting in many plants and (ii) more wilt incidence in flowering and podding stage, we wanted to estimate the loss in yield in relation to the stage at which wilt occurs. We now have 2-year data on loss in yield on a per plant basis.

The data are presented in Table 2.

Table 2. Grain yield loss in pigeonpea (cv. Sharda) as influenced by the stage at which wilt occurred[a]

Stage at which plants wilted	Yield per plant (g)	Actual loss of yield (g)	Loss of yield (%)	Normal seed weight (%)	Wrinkled seed weight (%)
Pre-pod	0.05	57.05	99.92		
Early pod	0.71	56.39	98.80	72.80	27.20
Pod-fill	6.35	50.75	88.85	86.01	13.99
Pod maturity	18.84	38.26	67.18	85.94	14.06
Pre-harvest	40.46	16.64	29.58	85.88	14.12
Healthy (check)	57.10	0.00	0.00	87.69	12.31

[a]Average grain yield from a total of 40 plants in 1976 and 1977 tests

It is clear that loss was almost complete when wilt occurred at or prior to early pod stage. Even when pods were full and plants close to harvest, the loss was around 30 percent in wilted plants. It is interesting to note that wilted plants produced over 70 percent normal seed and when the wilt was delayed, the percentage of normal seed produced was almost equal to the percentage produced on healthy plants. The test was carried out only on one cultivar; i.e., Sharda, and it is possible that other cultivars might show different loss patterns. However, we expect the general pattern would remain the same; i.e., lesser loss with late wilting.

5. Symptoms

When Butler published his paper in 1906, he described the symptoms fairly accurately. Very little addition to that description has been made since then. The infected plants show symptoms of gradual chlorosis and wilting starting from 4 to 6 seeks after planting. However, more wilt is observed during the flowering and podding stage. Black streaks in the vascular region as well as under the bark are characteristic.

Partial wilting in affected plants is not uncommon. Many such plants show a dark purple band extending from the base to several feet above ground towards wilted branches. We could often trace the band to one of the two major lateral roots of such a plant. Infection of the tap root most commonly produced complete wilting, whereas infection starting and extending from one of the two lateral roots more often caused partial wilting. Exceptions, however, were observed.

The dried leaves on wilted plants do not shed for a long time.

6. Morphological variation in the fungus

We made hundreds of isolations from specimens collected at Hyderabad and a large number of other locations visited during surveys. This species, like most other *Fusarium* spp., shows a great deal of variation in cultural characters. Based on characteristics such as type of growth, sporulation and colour and change in medium colour, we have classified these into 12 distinct groups (A to L). We are of course not the first to do this kind of work. Even Butler reported this type of work in 1910. Many other workers have done so since then (Sarojini, 1951; Subramaniam, 1955; Baldev and Amin, 1974).

We have not yet made any attempt to ascertain existence of physiologic races. Baldev and Amin (1974) presented evidence to suggest the existence of races. Their work, however, suffers from certain weaknesses. For example it is not clarified whether the three cultivars [NP(WR)-15, T-21, and C-11] which they used as differentials were homozygous for resistance to at least one isolate. It has been our experience that unless selfing is resorted to for several generations, the cultivars show considerable heterogeneity for different traits including disease reaction as a result of natural cross-pollination. Also the tests with different fungus isolates were carried out only once. In spite of this, we admit that the results presented by them do point to the possibility of the existence of races.

We have single-spored the 12 isolates, had the identification confirmed by the Commonwealth Mycological Institute, and have preserved them on autoclaved sand.

7. At what stage are plants infected?

As mentioned elsewhere, the disease incidence is very low in the first two months. More incidence is seen during flowering and podding stages. We, therefore, carried out a study to detect the fungus in the plants prior to the appearance of wilt symptoms. Plants of the susceptible cultivar, Sharda, grown in a wilt-sick plot, were used for this study. In 1977-78 season ten plants were removed 15, 30, and 45 days after sowing. In 1976-77 season, the fungus could be detected from collar region downwards in apparently healthy plants (3 to 5 plants only) collected 30 days after sowing, but not in those collected 15 days after sowing.

However, in 1977-78 season, the fungus could be detected in plants 15 days after sowing. The first wilted plant was noticed in the plot 45 days after sowing in 1976-77 and 30 days after sowing in 1977-78. This study shows that the plants are infected fairly early in the season and many plants apparently keep on 'fighting' the fungus until flowering/ podding.

While we were attempting to detect the infection prior to symptom appearance, through fungus isolation, we came across a paper by Miller-Jones et al. (1977) wherein they reported detection of infection of *Salix alba* var. *caerulea* (Cricket bat willow) by *Erwinia salicis*, before symptom appearance, by using an instrument called Shigometer. Diseased tissues were distinguished from healthy by their low resistance to a pulsed electric current. We got ICRISAT Electronics Engineer (Instrumentation), Mr. S.K.V.K. Chari, interested in the pigeonpea wilt problem. He has developed a similar instrument, using direct current, tentatively called by him as 'wilt detector'. Preliminary tests were carried out in pots as well as field. Plants were raised in sick soil. Electrical resistance was measured every 3 to 4 days. Plants showing a drop of more than 0.4 KΩ between two readings ultimately showed wilt. Work is being continued.

8. Systemicity of the fungus

The purpose of this study was mainly to confirm the findings of Mohonty (1949) who reported that the fungus was systemic. Five completely wilted plants of three cultivars (Sharda, BDN-1, ICP-6997) were selected and samples were taken for isolation every six inches from

root tip to the top and included leaflets, petioles, rachis, pedicel, pod hulls, flowers and seeds. The seed samples were collected after surface-sterilizing the pods with 0.1% mercuric chloride. The samples from individual plants were plated on modified Czapek's-Dox agar selective medium (Sharma and Singh, 1973) after surface sterilization with mercuric chloride. The plates were incubated at 28^{C} to 30^{O}C for 15 days. *Fusarium udum* was isolated from tap root, lateral roots, collar region, main stem, branches, leaflets, petioles, rachis, pedicel and pod hulls. However it could not be isolated from flowers or seeds.

Fusarium udum, however, can be detected as a surface contaminant on non surface-sterilized seed.

9. Survival

We have failed to find in the published literature any work done specifically to ascertain how long the fungus survives in wilted plant stubble. McRae and Shaw (1933) made the following statement:

> "Exposed in the open the fungus in many of the stems and roots dies but when kept in a cooler room in the shade most of it survives. The source of infection then exists in the uncut portions of roots below the ploughing-depth. From such parts of roots in situ the fungus has been isolated after two years though with difficulty, so even here it would appear that the fungus dies out though more slowly. Disinfected rahar (pigeonpea) seed sown in land free from a rahar crop for from eight to twenty years generally produces a crop with little or no wilt, while with a shorter interval the crop

comes up more or less severely wilted according to the shortness of the interval."

This indicates that the fungus survives something less than 8 years. Agnihotrudu (1954) has shown that *F. udum* does not colonize plant debris in the soil but can survive only in tissues already invaded as a pathogen. It then follows that the stubble fragments may be enabling the fungus to survive in soil up to 8 years. To find out how long *F. udum* survives in pigeonpea stubble an experiment was initiated in November 1974. Stubbles (root system with about 15-cm long stem base) of naturally infected plants were obtained, weighed, and buried in 35-cm diameter earthen pots. Two sets were prepared; one with black soil (vertisol) and the other with red soil (alfisol) collected from ICRISAT Center farm. Some properties of these two soils have been indicated in Table 3.

Table 3. Some properties of vertisol and alfisol used in the pigeonpea wilt fungus survival study

Soil type	pH (1:2)	E.C. mmho/cm	Organic carbon	Available P	Mechanical analysis		
					Sand %	Silt %	Clay %
Alfisol	5.90	0.10	0.20	2.10	59.60	7.20	33.2
Vertisol	7.85	0.15	0.38	1.60	38.80	20.00	41.2

Sixty pots, 30 with vertisol and 30 with alfisol, were prepared and buried in the ground so that the top of the pots was in line with the ground surface. Stubbles from six pots (3 vertisol + 3 alfisol) were removed after every six months, their weight taken and then checked for

the survival of *F. udum*. The experiment was planned for five years. Weather data (average max. and min. temperatures and rainfall) from Meteorological Station of ICRISAT were noted. The identity of the fungus was verified through microscopic observations and pathogenicity of some representative isolates was checked. In addition assistance from the Commonwealth Mycological Institute was sought. The data obtained after every 6-month interval have been given in detail in our annual reports of 1974-1978.

We were able to detect *F. udum* in stubble fragments from vertisol up to 2½ years and from alfisol up to 3 years. Based on this limited study, we are unable to understand how the fungus could survive up to eight years as suggested by McRae and Shaw (1933).

Some studies by other workers need to be mentioned in connection with the survival of *F. udum*. Sarojini (1950) concluded through pot studies that application of zinc (20, 40, and 80 ppm) to soil in which infected stubble were buried resulted in the disappearance of the fungus in 5 to 6 weeks. Boron and Manganese were less effective. Dey (1948) has claimed reduction in the wilt incidence when sorghum was grown as an intercrop. Bose (1939) made a chance observation of reduced wilt incidence in a field where tobacco was grown in the preceding season. McRae and Shaw (1933) through observations in permanent manurial and rotation experiments over several years reported (i) manuring with superphosphate (7-23 lb P_2O_5/acre) and with cattle manure increased the wilt, (ii) green manuring with *Crotalaria juncea* (60 lb seed/acre) decreased wilt, and (iii) superphosphate and green manure together increased wilt.

10. Screening techniques

Since one of the major objectives of our program is to assist the breeders in developing disease resistant varieties, we have spent a great deal of our time in working out efficient and simple techniques to screen germplasm and breeding material for resistance to different diseases including pigeonpea wilt.

(i) Water culture

The technique essentially consists of transplanting pigeonpea seedlings, raised in autoclaved sand, into glass tubes containing aqueous suspension of *F. udum* conidia. We spent a great deal of time in developing this technique but gave it up subsequently because of the lack of correlation between the results obtained by this technique with those of field screening results. The same technique works well in case of chickpea wilt and therefore we shall give more details elsewhere.

When we first developed this technique, we thought we had worked out something original. Subsequently we discovered that similar techniques had been described by Wensley and McKeen (1962) and Roberts and Kraft (1971). We were, however, surprised to note that the idea of such a technique had occurred to Butler (1910). He used water culture (he called it so) for studying the site of root infection. Who knows, we may discover an even earlier reference to this technique!

(ii) Pot screening

The well-known technique of transplanting seedlings of which roots are injured and inoculated to autoclaved sand/soil in pots gave us erratic results. On the other hand we had good success in preliminary tests with the following procedure:

1. Alfisol (non-autoclaved) is filled in large (35-cm) earthen pots.
2. *Fusarium udum* is multiplied on sand-pigeonpea flour (9:1) medium (SPM) for 15 days.
3. Fungus on SPM (200 g) and autoclaved pigeonpea stem bits (200 g) are mixed with the top 15-cm of soil in pots.
4. Susceptible cultivar ICP-6997 (approx. 50 seeds) is raised in each pot. All plants wilted within 60 days are chopped and incorporated in the same pot.
5. Step 3 given above repeated.
6. Step 4 given above repeated.
7. Step 4 repeated once more.

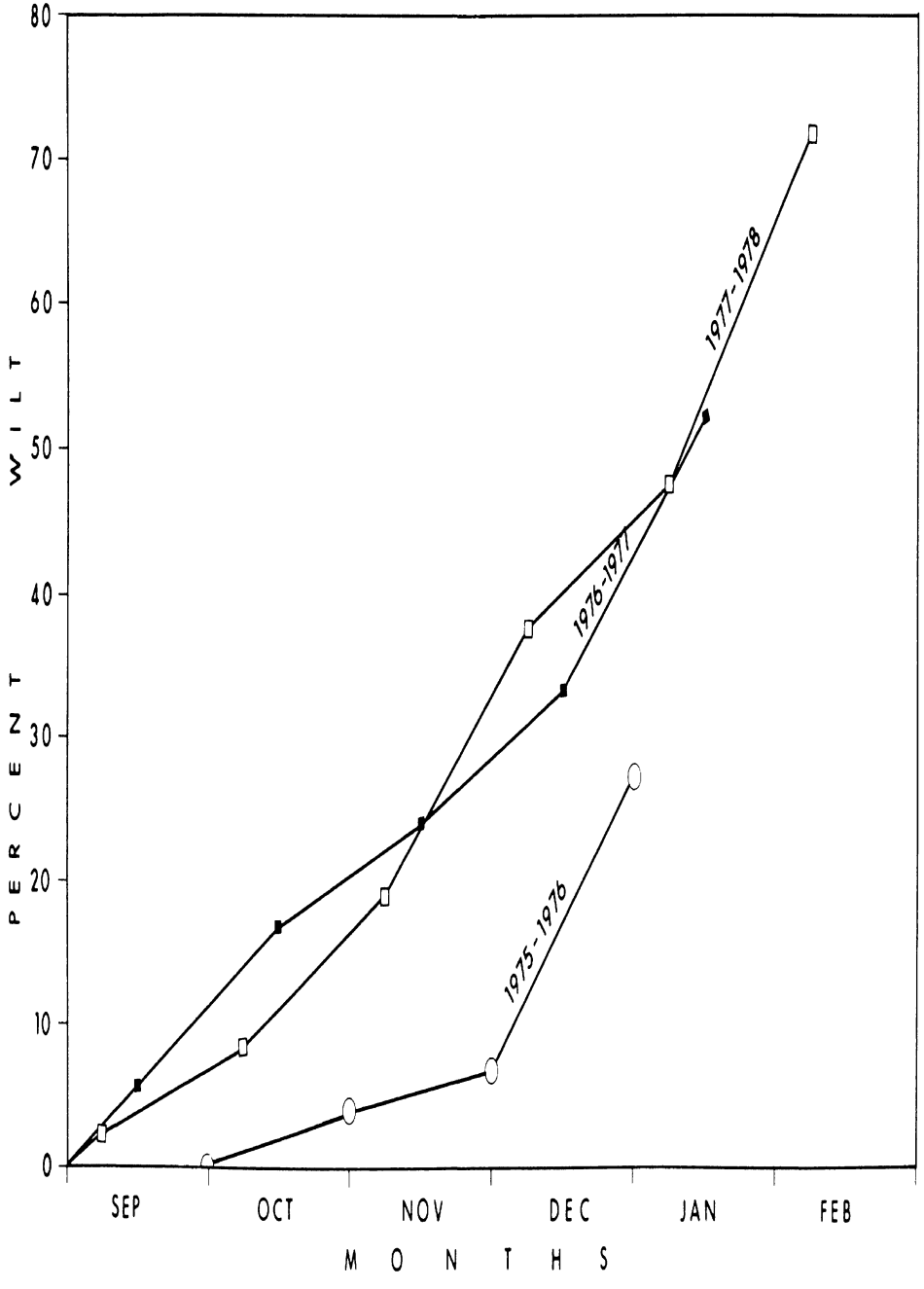
After step 7 we get over 90 percent wilt in each pot. Currently we are developing 1,000 such pots mainly to have a screening procedure to support field screening.

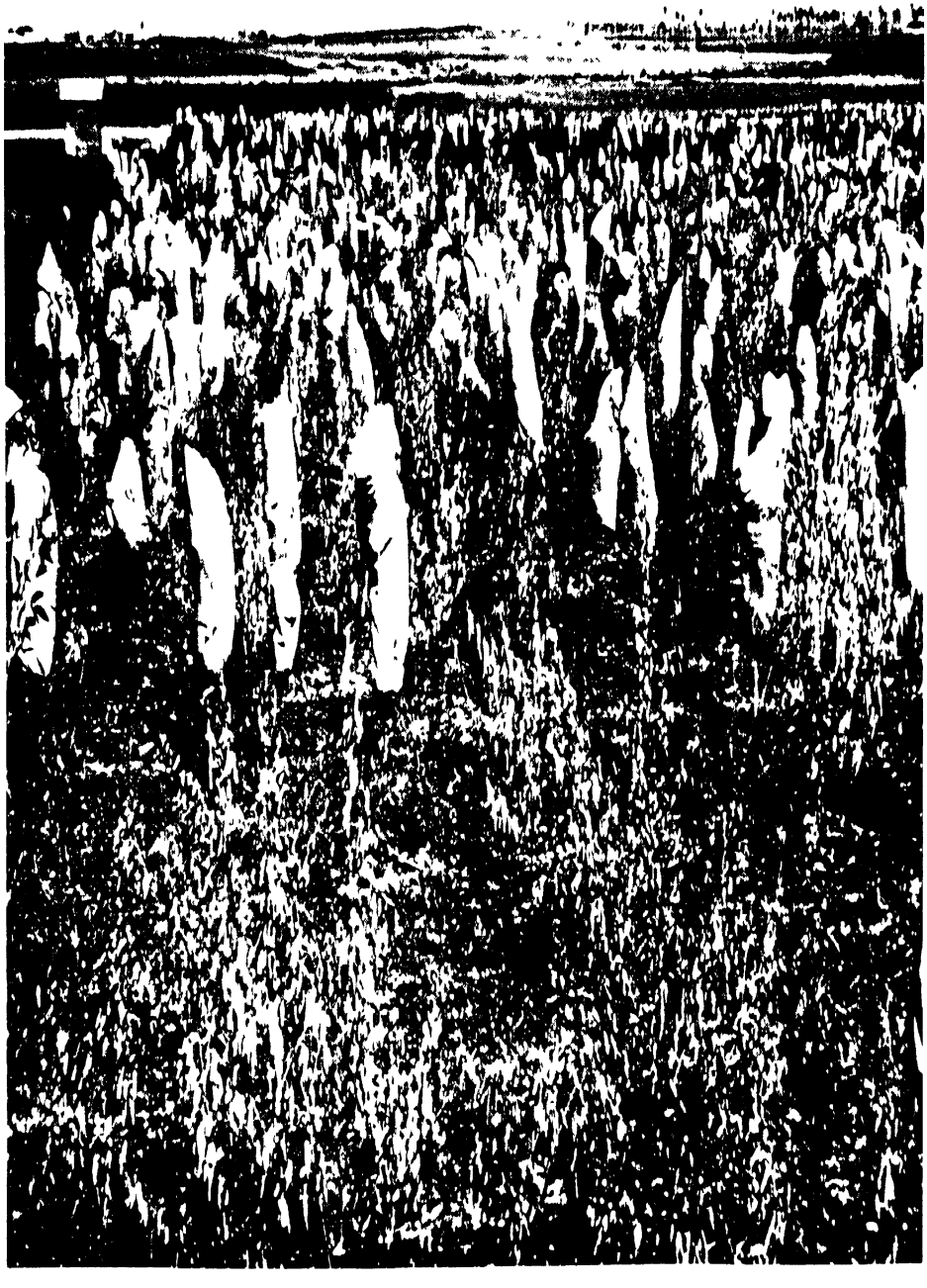
We have yet to verify the success of this technique.

(iii) Sick plot

The idea of using a sick plot is well-known and this procedure has been used for a long time for screening against several vascular wilts. We have developed two sick plots in vertisol (1.5 ha each) and two small sick plots in alfisol (0.1 ha and 0.4 ha). Fig.1 gives an idea as to how the "sickness" has developed in one of the vertisol plots over three seasons. Our experience tells us that "sickness" develops more quickly in alfisol than in vertisol. Also wilt shows up earlier in alfisol than in vertisol. It is pertinent to point out here the pot

FIG. 1 MONTH-WISE PIGEONPEA (C.V. SHARDA) WILT INCIDENCE IN SICK PLOT 'A' DURING 1975-1976, 1976-1977 AND 1977-1978





PIGEONPEA WILT SCREENING IN A SICK PLOT AT HYDERABAD

studies of Shukla (1975) which revealed that the wilt incidence was high in sand alone (93.75%) and least in heavy black soil (18.18%). The disease increased with the decrease in the proportion of soil in soil-sand mixture.

The procedures we followed in developing wilt-sick plots have been given in Appendix-I. At first we multiplied the fungus on materials other than pigeonpea stubble, but later realized that the best way is to incorporate the stubble from diseased plants and grow wilt susceptible cultivars in intermittent rows all over the field.

The planting pattern we are following for screening is one susceptible check row after every two test rows in plots which are in the process of becoming 'sick' and one susceptible check row after every four test rows in plots which have already become 'sick'.

11. Screening work done so far

Screening work was initiated in India from the time the disease was described in 1906. Research centers where resistance work was or is being carried out are: Poona (Butler,1910), Pusa (McRae and Shaw, 1933), Delhi (Deshpande et al. 1963), Kanpur (Dey, 1948), Parbhani (Raut and Bhombe, 1971), Sangareddy, Hyderabad (Vaheeduddin, 1958), and of course now ICRISAT. Several cultivars have been claimed resistant. When we tested many of these, we did not get uniformity in performance. It is possible that the seed which we have in our germplasm collection came from outcrossed plants and therefore many plants show susceptibility. Some of the cultivars which consistently show low disease level are NP(WR)-15 (N.P.24 x N.P.51), 15-3-3, BDN-1, and 20-1. Another cultivar NP-80 is

mentioned repeatedly in the literature since 1933 (McRae and Shaw, 1933) as a highly resistant one. The seed of N.P.80 has, however, not been available to us for testing.

Since it took some time to develop a good sick plot, we could initiate dependable field screening only in the 1976-77 season. As the first step we focussed our attention on (i) already claimed resistant cultivars and (ii) lines identified as resistant to another important disease, sterility mosaic. We have been discarding the susceptible segregants and selfing individual resistant plants to fix wilt resistance in a homozygous condition. We now have some promising lines which come from both types of materials indicated above. Systematic screening of germplasm has been initiated but has been given low priority at this time. Screening of breeding populations generated by ICRISAT breeders is being carried out. Multilication testing of promising lines has been initiated. Table 4 summarizes ICRISAT's screening work.

Table 4. Screening for resistance to pigeonpea wilt at ICRISAT

Materials screened in 1976-77 and 1977-78	
Breeding materials	2,000
Germplasm	194
Promising lines identified	19
Under multilocation test	12
Promising against wilt and sterility mosaic	10
Breeding materials being screened in 1978-79	4,000

12. Resistant/tolerant lines

At this stage we feel reasonably confident about the performance of the following lines when grown as annuals (no ratoon crop). Some of these are resistant to sterility mosaic also (marked*).

ICP-8859, -8860, -8861*, -8862*, -8863, -8864,

-8865, -8867*, -8868, and -8869*

It may be pointed out that most of these are still apparently segregating, giving a very small percentage of susceptible segregants. We are continuing to self single plants and advance their progenies to the next season.

Most pigeonpea cultivars have a tendency towards being perennials. Therefore after the first harvest of pods, the plants produce new leaves and another flush of flowers/pods (ratoon crop). We find that all the promising lines indicated above show high wilt incidence in the first ratoon itself. We have been able to detect the presence of the fungus in many of these lines before the first harvest. Apparently the fungus is held in check by these lines until the first harvest is over, after which the fungus dominates and kills the plants.

II. Phytophthora Blight

1. Earlier work

A 'stem rot of pigeonpea' was described for the first time from India by Pal et al. in 1970, although its suspected occurrence was reported by Williams et al. (1966). These workers observed the disease in serious form in the 1968-69 season at certain locations in northern India. The causal fungus was identified as *Phytophthora drechsleri*

Tucker var. *cajani* Pal, Grewal and Sarbhoy. Five years later a 'Phytophthora stem blight' of pigeonpea was described from the same areas of northern India (Williams et al. 1975). The species was not identified at that time, but was later described by the same group of workers as *Phytophthora cajani* (Amin et al. 1978)

2. Occurrence

The disease has been reported from the northern Indian states of Delhi and Uttar Pradesh. A similar disease was observed by us at ICRISAT Center in 1976 in severe form. Although we have not conducted extensive surveys, we suspect the disease occurs in most pigeonpea growing areas, particularly during longer wet spells which are common during the first three months of crop growth. Information on losses caused by this disease is not available, but there is no doubt that the disease has the potential to cause devastation in a susceptible cultivar. One of us (YLN) was told during his trip to central America in November 1977 that Phytophthora stem blight incidence is commonly observed in Puerto Rico, Dominican Republic, and Trinidad. *P. parasitica* was mentioned as the species affecting pigeonpea in Puerto Rico.

3. Symptoms

The symptoms have been described by Pal et al. (1970) and Williams et al. (1975). The symptoms can be seen only on above-ground parts, and the root system as well as the portion of the stem below the soil surface are not affected. The description given by Williams et al. (1975) is reproduced on the next page.

"Symptoms include rapid wilting of the plant parts above the invasion site; dessication and upward rolling of leaflets, usually without chlorosis; withering of petioles and small stems; and dark-brown to black necrotic lesions encircling the stem at the base, or up to a meter or more above soil level. Lesions at the plant base often extend 15-20 cm up the stem. Lesions on the upper parts of the plant are on the main stem, branches, or petioles, usually have definite margins, and initially have a plane surface which later becomes slightly depressed. Lesions are often centered on a leaf scar, and extend several centimeters in each direction from the apparent invasion site. Longitudinal cuts into newly formed lesions show brown-to-black discoloration of the bark and cambium, but not the older xylem. Later, the older xylem tissue may become discolored and the stem may break at the lesion site. Gross symptoms resemble those of Fusarium wilt (caused by *Fusarium udum* Butler), and it is possible that Phytophthora stem blight has been confused with this disease in the past."

In addition to the above symptoms, we have observed at ICRISAT Center water-soaked lesions on leaves from which the fungus can be isolated.

4. Identification of species

Since we could not identify the species isolated at ICRISAT Center, we sought help from the Commonwealth Mycological Institute, U.K. for expert opinion. Dr. D. J. Stamps identified the species as *Phytophthora vignae* (IMI-211490). When we attempted to obtain infection of cowpea

(11 cvs.; viz., var.57, 1149, 1160, G.C.187, G.C.10-72, var.25/3/2, Sel.K-1, FS-68, New Era, Pale Green, and Pusa Dofasli) with the fungus, we failed in repeated tests. We, therefore, took up the question with Dr. Stamps. Her comments are reproduced below:

> ".....morphological features agreed more closely with those described for *P. vignae*, though we have no type culture here for comparison. However, in view of the difference in pathogenicity now known, identification with *P. vignae* should perhaps be reconsidered."

A comparison of our *Phytophthora* with other species was made by us in 1976-77. Table 5 has been reproduced from our annual report of 1976-77.

One of us (JK) is currently (October 15 to December 15, 1978) working with Dr. D. C. Erwin at the University California, Riverside, California, USA and hopefully we should be able to know soon what species of *Phytophthora* is involved in causing blight at ICRISAT Center.

We must emphasize here that the symptoms we observe at ICRISAT Center are identical to those that are seen in diseased plants in Delhi and Uttar Pradesh states in northern India.

5. Survival

There is no published material related to this topic. We have yet to initiate extensive studies. However, we wish to record a few observations.

(i) We have seen the disease in fields where pigeonpea had not been cultivated at least for the preceding four years.

Table 5. Comparison of the characters of pigeonpea *Phytophthora*

Characters	*P. drechsleri* var. *cajani* (Mahendra Pal et al. 1970)	*Phytophthora* sp. (Williams et al. 1975)	*P. vignae* Purss[a] (1963)	*Phytophthora* sp. (ICRISAT, 1976)
1	2	3	4	5
1. Hyphal swellings	Not mentioned	Not present	Present	Present
2. Sporangia	Ovate to pyriform and very few spherical 9-33 x 4.7-13.9μ Av.17.4-22 x 8.0-11.6μ with a minute papilla	Ovoid to obpyriform 49-82μ (Av.60μ), terminal, persistent and non-papillate	Ellipsoid, ovoid or obpyriform often tapering somewhat to the base Av.48 x 27 (max.72x54)μ non-papillate apical thickening inconspicuous	Ovate to pyriform 10,0-27.5 x 7.5-17.5μ (18.4 x 11.0)μ mostly non-papillate
3. Zoospores	8 to 20 in number in each sporangium, and sometimes they liberate out with an evanascent type of vesicle or proliferation of zoosporangium	Zoospores differentiated within the sporangium and were released one by one upon the dehiscene of sporangial apex	Not mentioned	-

contd.

1	2	3	4	5
4. Sex organs	*Oogonia* spherical 23.4-37.0μ, amphigynous *antheridia* nearly spherical (8.1-15.0x8-14μ) Av.(11.6x12.7μ)	*Oogonia* with amphigynous *antheridia* were formed on the same hyphae	*Oogonia* **spherical** 32 (max.46)μ dia, *antheridia* all amphigynous, variable in size and shape Av.16 x 15 (max. 27 x 18)μ	*Oogonia* with amphigynous *antheridia* were formed.
	Oospores spherical to globose 23.4-37μ (Av.30μ) in dia.	Oospores single, spherical, light brown, smooth, and plerotic. Because of intermediate exit pore, 6.6-10μm the present sp. does not fit into any of the six groups of Waterhouse.	Oospores loose in the oogonium 26 (max.32)μ dia.	Oospores spherical, 27.5-47.5μ (37.3μ) in dia.
5. Optimum temperature for the growth	30-32°C	30°C	28-30°C	Approx. 30°C

contd.

1	2	3	4	5
6. Host range	Not tested	Non-hosts: Green gram, Black gram, beans, soybean, cowpea, chickpea, safflower, *Xanthium*, *Cannabis*, *Croton*, and *Atylosia scarabaeoides*	Not given. However it has been reported on cowpea.	Non-hosts: Green gram, Black gram, French bean, Lima bean, cowpea (11 cvs.), chickpea
7. Chlamydospores	Present	Not present	Not present	Not present

[a]Commonwealth Mycological Institute, Mycol. Paper No.92, p.17, 1963.

(ii) In seed pathology studies, we have so far not observed any *Phytophthora*.

(iii) Artificial inoculations of several plant species other than pigeonpea have been unsuccessful.

(iv) In general more disease is seen in pigeonpea grown in alfisol than in vertisol.

(v) More disease incidence is observed in low-lying patches. In poorly drained fields, an increase in the disease is seen in successive pigeonpea crops, whereas the disease may not show at all in a similar cropping situation in well-drained soil.

(vi) Infected stem bits when left on the surface of soil in pots (kept in the open) failed to provide inoculum to infect the susceptible cv. HY-3C after four months (This was a preliminary study).

(vii) We have been able to detect oospores in diseased leaves.

6. Screening techniques

(i) Pot screening

We have been able to standardize a pot screening procedure. The steps followed are:

1. Isolate P_2 of *Phytophthora* sp. isolated at ICRISAT Center is grown on V-8 juice agar (V-8 juice-100 ml; $CaCO_3$-2 g; agar-20 g; distilled water-900 ml) for one week (28^{o}-30^{o}C).

Pot screening for Phytophthora Blight resistance. Note some pots having no seedling mortality.

2. Five mm discs of the culture are transferred to 100 ml autoclaved V-8 juice broth (as above without agar) in 250 ml flasks. Incubation is at 28°-30°C for 15 days.
3. The mycelial mat from each flask is removed and washed twice with distilled water. It is then macerated in 100 ml distilled water in a Waring blendor for 2 to 3 min. The suspension prepared this way serves as inoculum.
4. Five to 10-day old seedlings (25-30), raised in non-sterilized alfisol (7.5 kg/pot) in 20-cm pots are inoculated by pouring 50 ml inoculum (step 3) diluted further with 50 ml of tap water (i.e., 100 ml inoculum per pot).
5. Susceptible checks (cv. HY-3C), both inoculated and non-inoculated, are kept with each batch of germplasm or breeding material.
6. Pots are liberally watered three times a day.
7. Symptoms usually appear in 48 hours. Final observation is taken 10 days after inoculation.

The above procedure has worked extremely satisfactorily and excellent correlation between pot and field screening has been observed.

(ii) Field screening

The steps followed are:

1. Isolate P_2 of *Phytophthora* sp. is grown in V-8 juice agar for one week (28°-30°C).
2. Inoculum is mixed well with medium after adding carborundum (600-mesh).

PIGEONPEA PHYTOPHTHORA BLIGHT AT ICRISAT.
TOLERANT CULTIVAR IN BACKGROUND

3. Individual plants (one month old) are inoculated at the collar region by rubbing.
4. The field is flood irrigated immediately afterwards and again one week later. The second irrigation is given only if dry weather prevails.
5. Typical blight symptoms appear within 10 days.
6. Surviving plants are reinoculated as above.

The method has worked satisfactorily, but we do find a small percentage of escapes. Also it is not the most convenient method. We are considering alternatives which will give us a more efficient and simpler technique.

7. Screening work

We initiated systematic screening work in the 1976-77 season. Table 6 summarizes the work.

Table 6. Summary of the work on screening pigeonpea for resistance to Phytophthora blight

Germplasm screened		
Pot	:	1,200
Field	:	343
Resistant lines identified	:	28
Breeding materials		
Screened	:	150
Being screened in 1978-79 season	:	1,700

8. Resistant lines

As mentioned in Table 6, we have identified 28 lines/cultivars resistant to the blight. These are: ICP-28, -113, -214, -231, -339, -580, -752, -913, -914, -934, -1088, -1090, -1120, -1123, -1149, -1150, -1151, -1258, -1321, -1529, -1535, -1570, -1950, -2376, -3753, -6974, -7065, -7182.

Atylosia is a wild relative of pigeonpea. Two species; *A. sericea* and *A. platycarpa*, have been found resistant.

9. Existence of physiologic races

When we subjected all the above 28 lines resistant to the ICRISAT isolate of *Phytophthora* to inoculations with an isolate from Kanpur, we found all of them to be susceptible. An isolate from New Delhi caused mortality of a certain percentage in each of the 28 lines. Once the identification of the *Phytophthora* species is settled, it would be possible to state whether the susceptibility of lines resistant to ICRISAT isolate to Kanpur and New Delhi isolates is due to a different species or due to a different race or races of the same species.

10. Chemical control

A newer fungicide Ridomil (Methyl 2(N(2-methoxyacetyl)-2, 6-xylidino) propionate) of CIBA has been found extremely effective against several diseases caused by phycomycetes. We have initiated studies on the control of *Phytophthora* through seed dressing in pot culture. The results are awaited.

III. Other Pathogens

Under certain situations we do find some other soil fungi causing problems in pigeonpea.

1. *Sclerotium rolfsii*

Seedling mortality caused by this fungus is fairly common in India and some other pigeonpea growing countries. We have observed more mortality when undecomposed stubble of cereals (e.g. sorghum) are present in the soil. One of the common practices at ICRISAT (in spite of our protest) is to chop and incorporate cereal stubble only a few days before planting pigeonpea. This practice, we feel, is mainly responsible for more seedling mortality caused by *Sclerotium rolfsii*.

2. *Rhizoctonia bataticola*

Dry root rot has been reported so far only from India. It is a minor problem in the normal season (June-December/March) crop, but a major problem when an off-season summer crop is attempted especially in black soils. One of the ways by which pigeonpea production in central/southern India can be increased is to have an extra crop between November-April. However, *R. bataticola* seriously hits this crop and we need to identify resistant genotypes if the idea of an extra crop is to succeed.

3. *Rhizoctonia solani*

Root rot in seedlings or aerial blight by this fungus has been reported/observed in India, Sierra Leone, Phillippines, and Malaysia. One of us (YLN) has observed serious aerial blight in experimental plantings in Malaysia. On the whole, however, it is a minor problem.

CHICKPEA (Bengal gram, gram, garbanzo, etc.)

I. Wilt Complex

1. History

Chickpea wilt was first mentioned by Butler in his book in 1918. In 1923 McKerral, working in Burma, considered the disease to be soil-borne. He sent specimens to India which yielded *Fusarium* sp. Narsimhan in 1929 reported association of *Fusarium* sp. and *Rhizoctonia* sp. with wilted plants. Later Dastur (1935) found *Rhizoctonia bataticola* producing 'wilted' plants and he called the disease 'Rhizoctonia wilt'. Although he isolated *Fusarium* from several wilted plants, he could not produce the disease artificially. In view of the fact that his description of symptoms (he did not look for vascular discoloration) and field pattern of incidence is almost identical to that of typical wilt caused by *Fusarium oxysporum* f. sp. *ciceri*, his failure to prove pathogenicity of the *Fusarium* he isolated is a mystery to us. He concluded that the wilt was due to 'physiological' reasons and called it 'physiological wilt'. In 1939 Prasad and Padwick published a detailed account of their studies and reported *Fusarium* sp. to be the cause of chickpea wilt. The fungus was named later by Padwick (1940) as *F. orthoceras* var. *ciceri*. Erwin (1958) from U.S.A. reported *F. lateritium* f. *ciceri* to be the cause and questioned the name *F. orthoceras* var. *ciceri*. Following the classification of Snyder and Hanson (1940), Chattopadhyay and Sen Gupta (1967) renamed *F. orthoceras* var. *ciceri* as *F. oxysporum* f. sp. *ciceri*. This change has been accepted by Booth (1971).

While on the one hand chickpea wilt was considered to be caused by *Fusarium*, on the other several workers were not convinced. In addition

to other fungi reportedly found associated with wilt, high temperatures at the time of sowing and flowering, deficient soil moisture and 'bad soil' were considered to be the causes (Bedi and Pracer, 1952; Anonymous, 1953). The state of Punjab in India had a project on chickpea wilt from 1947-1954 (J.S. Chohan - personal communication) and it was concluded that soil and weather factors, and not fungi, were the cause. It seems that the use of the term 'wilt complex' began after all these investigations and any dead/dried chickpea plant was considered wilted due to 'wilt complex'. A report on virus-induced wilts in chickpea from Iran (Kaiser and Danesh, 1971) further contributed to the confusion in India. In the literature we find the term 'wilt' used loosely for root rots and even blights. So much confusion has existed since then that it prompted Dr. H.K. Jain, now Director of the Indian Agricultural Research Institute, New Delhi, to organize a symposium in 1973 on "Problems of wilt and breeding for wilt resistance in Bengal gram". Several Indian pathologists and breeders participated and a part of one of the conclusions reproduced below (Jain and Bahl, 1974) pointed out the problem clearly:

> "The participants concluded that considerable confusion exists with regard to the causation of the wilt disease of Bengal gram, most workers have tended to emphasize a wide variety of factors including those of physiological, agronomical, environmental and pathological nature, which in one way or the other contribute to the development of wilt symptoms."

This was the status of the problem when we initiated our investigations at ICRISAT. It was clear that various causal agents were responsible for the drying of plants and the foremost need was to understand the

characteristic symptoms produced by each. Once the diagnosis of the cause based on host symptoms became possible, there would be no room for confusion.

We have gone into details above mainly to ensure a proper understanding of the problem itself and the reason why we devoted considerable time to investigate the so-called "wilt complex". Although the term "wilt complex" has been used mainly in India, we have noted through literature similar situations in some other chickpea growing countries.

2. ICRISAT work

We initiated a project in 1974 to understand the "wilt complex". After many critical observations of symptoms, hundreds of isolations of fungi in pure cultures, pathogenicity tests, and visits to research stations and farmers' fields in India and other chickpea growing countries, we concluded that what has generally been referred as the "wilt complex" is actually a number of distinct diagnosable diseases. In order to assist workers in identifying the main disorders of chickpea, we have prepared a bulletin with colored plates (in press). We have made an attempt to develop a key to diagnose the common, but confusing, disorders. The key from the bulletin is reproduced on the next page.

Key for the diagnosis of wilt-like disorders of chickpea

CHICKPEA PLANTS SHOWING PREMATURE WILTING/DRYING

I. Wilting (drooping of petiole and rachis)

A. No external root rot

1. Internal (xylem) discolora- ... *Fusarium oxysporum* f. sp. *ciceri* (WILT)
   tion

2. No internal discoloration; ... Frost injury (to be confirmed through weather data)
   irregular pattern of leaflet scorching

B. External root rot (tap root not brittle)

1. Rotting at collar region downwards; small (1 mm), brown, round, rapeseed-like sclerotia visible at base along with white mycelium ... *Sclerotium rolfsii* (COLLAR ROT)

2. Dark brown lesion extending on stem above collar region; lesion can extend to lower branches; no sclerotia seen ... *Rhizoctonia solani* (ROOT ROT)

3. Dark brown lesion at base mycelium not visible; internal brown discoloration restricted to periphery of the wood ... *Operculella padwickii* (FOOT ROT)

C. External base/stem lesion; white mycelium on lesions with/without white mycelial knots developing into dark sclerotia ... *Sclerotinia sclerotiorum* (STEM ROT)

II. Drying without general wilting

A. Stunting/discoloration

1. No external rotting of roots

a) Proliferation of branches

i) Browning of leaves in desi and yellowing in kabuli cultivars; phloem necrosis in the collar region ... Unidentified virus (STUNT)

ii) Terminal bud necrosis mild mottle clearly seen on broader leaflets of kabuli cultivars; no phloem necrosis ... Alfalfa Mosaic Virus (MOSAIC)

b) No proliferation of branches

i) Browning of older leaflets in desi and yellowing in kabuli cultivars; younger leaflets remain green; no phloem browning ... Salinity injury

ii) Young foliage bright yellow; terminal bud necrosis; mottle at mid-height on a recovering plant ... Iron deficiency (CHLOROSIS)

2. External rotting of roots; galls on roots quite distinct from *Rhizobium* nodules ... *Meloidogyne* spp. (ROOT-KNOT)

B. No stunting/discoloration; only tops may show drooping; rotting of most roots; tap root brittle; minute sclerotia and/or sparse grey mycelium in pith cavity in the collar region, which can be seen with a 10X hand lens. Also the sclerotia can be seen under the root bark which peels off easily.	...	*Rhizoctonia bataticola* (DRY ROOT ROT)

We wish to make a special mention of chickpea stunt. We feel that this particular disease, which is observed at most places in India and also many other chickpea growing countries, contributed in a major way to the confusion in diagnosis. Very frequently it is possible to isolate *Fusarium* spp. from the root system of the stunt affected plants, but no one could produce typical stunt symptoms with any *Fusarium*. It is pertinent to cite here the observations made by Prasad and Padwick (1939). They divided the wilt affected plants into three groups on the basis of symptoms. These were:

"1. Those in which the first symptom was drooping of the upper leaves followed soon by the lower leaves. The plants withered and died within about a week.

2. Those in which the leaves gradually turned yellow and then began to drop, the remaining leaves rapidly withering and the plant dying.

3. Those in which the leaves became red. In the later stages these plants resembled those of group (2)."

Whereas the symptoms of first group above are of typical wilt (*Fusarium oxysporum* f. sp. *ciceri*), the symptoms in the second group can also be seen in the wilt in certain genotypes. The symptoms of the third group, however, are never seen in wilt and we feel certain that those are of stunt. Further Prasad and Padwick (1939) mentioned phloem browning as a symptom of wilt, but in the results of their pathogenicity tests they did not mention red leaves or phloem browning. Obviously they were unable to produce those symptoms through inoculations with *Fusarium*. It seems, therefore, that chickpea stunt was not identified earlier and was confusing the workers.

II. Wilt (*Fusarium oxysporum* f. sp. *ciceri*)

1. Occurrence

The disease is relatively more serious and has been reported from Burma, India, Mexico, Pakistan, Peru and U.S.A. From several other countries, *Fusarium* species have been reported and we presume that the wilt fungus is also present in those countries. The disease is widely prevalent in India.

2. Symptoms

We have given a detailed description of symptoms in the bulletin (in press) for diagnosing wilt-like disorders of chickpea. The characteristic symptoms are (i) sudden drooping of leaves and petioles, (ii) no external rotting of roots, and (iii) black internal discoloration involving xylem and pith.

3. Early/late wilt

In northern India wilt is often referred to as 'early' or 'late' wilt depending upon the time of occurrence. Early wilt refers to seedling wilt (October-November) and late wilt refers to wilting at post-flowering stage (February-March). Generally the wilt incidence is negligible in the intervening period. We think it is possibly due to the cold winter in northern India that the wilt incidence is negligible during the vegetative stage. With moderate winter at Hyderabad, we have not noticed any clear-cut 'early' or 'late' wilt; in fact wilt occurs here right from the seedling through the podding stage.

4. Loss estimation

As in several other diseases, no precise information on losses caused by this disease is available from any country. According to a rough estimate about 10 percent loss in yield due to wilt was considered to be a regular feature in chickpea growing states of India (Singh and Dahiya, 1973). According to Grewal et al. (1974), 2 to 5 percent loss is caused every year in India, but it could go as high as 60 percent. In both these reports the term wilt was used in a general sense to include mortality due to various causes, and not due to only *F. oxysporum* f. sp.*ciceri*.

To get an idea about the loss on a per plant basis in relation to the stage at which the wilt occurs, we conducted an experiment in the 1977-78 season. Wilting prior to the flowering stage of course results in total loss. We, therefore, selected stages after podding had begun. Four cultivars were included in the study. These were sown on

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October 14, 1977 in a wilt-sick plot and also in a nonwilt-sick plot. Healthy plants were obtained from the latter as most of the plants of these cultivars in the wilt-sick plot were affected. Thirty plants of cvs. Chafa, P-436, JG-62 and 850-3/27 showing wilt at three stages (flowering/podding; full podding; pre-harvest) were tagged from January 15 onwards and harvested on February 27, 1978. Likewise 30 healthy plants of each cultivar were also harvested for comparing yields and estimating losses.

The data on grain yield loss and loss in 100-seed weight is presented in Table 7.

The data presented in Table 7 reveal (i) earlier wilting caused more loss than late wilting, though even the latter resulted in substantial loss, (ii) the 100-seed weight was adversely affected by wilt, and (iii) loss in seed weight at all the three stages of wilting was much more in JG-62 and P-436 than in Chafa and 850-3/27.

Seeds harvested from diseased plants of chickpea were lighter, rough (wrinkled surface) and dull in colour as compared to healthy ones.

Chauhan (1960) attempted to develop a loss estimation technique based on the time and amount of wilting. There was, however, no follow-up on that.

5. At what stage are plants infected?

We conducted experiments in 1977-78 season to get an answer to the above question. Two cultivars, one highly susceptible (JG-62) and one moderately susceptible (850-3/27), were raised in heavily inoculated soil in pots. Whereas cv. JG-62 was infected on the fourth day after

Table 7. Influence of wilting at different stages on the grain yield of four chickpea cultivars[a]

Cultivar	Stage of plant[b]	Average number of seeds/ plant	Average seed weight/ plant (g)	Percent loss in seed weight/ plant	100-seed weight (g)	Percent reduction in 100-seed weight
Chafa						
(Wilted)	S1	22	2.80	89.23	13.09	22.12
	S2	60	7.85	69.80	14.00	16.71
	S3	132	19.86	23.61	15.00	10.76
(Healthy)		158	26.00		16.81	
P-436						
(Wilted)	S1	25	2.08	91.40	9.44	35.16
	S2	56	5.66	76.61	10.37	28.77
	S3	121	12.16	49.75	11.17	23.28
(healthy)		161	24.20		14.56	
JG-62						
(Wilted)	S1	15	1.44	94.26	8.44	44.51
	S2	42	4.36	82.65	9.62	36.75
	S3	133	14.76	41.26	12.18	19.92
(Healthy)		166	25.13		15.21	
850-3/27						
(Wilted)	S1	9	1.41	91.45	15.75	43.44
	S2	20	5.83	64.66	20.85	25.13
	S3	50	12.10	26.66	23.31	16.30
(Healthy)		61	16.50		27.85	

[a]Data represent averages of 30 plants.
[b]S1 - Flowering and podding
S2 - Full podding
S3 - Pre-harvest

sowing, the cv. 850-3/27 was infected on the seventh day. JG-62 showed 100 percent infection within six days but 850-3/27 showed that much

in 20 days. Age of chickpea plants at the time of inoculation was found to influence infection. Cultivars JG-62 and 850-3/27 could not be infected after they reached the age 70 and 63 days, respectively.

6. Systemicity

In repeated studies we have confirmed that the fungus is systemic and can be isolated from all parts of an infected plant including the seed.

7. Seed transmission

Our studies have conclusively established that the fungus can be internally seed-borne and it is located mostly as chlamydospores in the hilum region of seed. Cultivars show differences in seed transmission percentage.

We have further found a fungicidal seed treatment to eradicate the fungus. Benlate-T (30% benomyl + 30% thiram) at 0.15% rate eradicates the fungus completely.

We have adapted a seed-clearing technique (using NaOH) to directly observe the fungus in the hilum region of seed.

It may be pointed out here that Erwin and Snyder (1958) had suspected seed transmission of the wilt fungus, but Westerlund et al. (1974) failed to obtain evidence of such transmission. It is not clear from the paper of Westerlund et al. (1974) whether the seeds they used for their tests were obtained from wilted plants. Likewise the name of the cultivar from which the seed was obtained was not mentioned. As pointed out earlier, it is important to know the cultivar, as there seem to be clear differences between cultivars with regard to percentage

seed transmission. In our tests we found that the extent of seed transmission in cv. Chafa was considerably less than in cvs. JG-62 and P-436.

8. Survival/host range

We have not seen any published paper on this aspect. It is logically presumed that the fungus survives in the dead plant debris in the soil. There are many questions related to this aspect which need answers. As a first step we have initiated an experiment to find out how long the fungus can be detected in dead plant tissue buried in the soil. The experiment is continuing. The fungus could be detected in the buried roots after six months. In leaflets and stem pieces, it could not be detected after 2 and 4 months, respectively.

Since nonsusceptible plant species are known to be carriers of pathogenic Fusaria (Armstrong and Armstrong, 1948) we wanted to know if such a situation exists in case of chickpea wilt *Fusarium*.

Plant species were sown in the chickpea wilt-sick plot in 5-m rows (50 seeds/row) along with the susceptible chickpea cv. JG-62 on October 28, 1977. They were observed for wilt symptoms up to March 1978. Isolations of *Fusarium* were attempted from five plants of each crop at 30-day intervals during the season. The results are presented in Table 8.

From wilt-sick plots, naturally growing weeds were collected throughout the season and isolations of *Fusarium* were attempted on a selective medium. The results are presented in Table 9.

Table 8. Detection of *Fusarium* in the roots of different plant species grown in the wilt-sick plot (B-5)

Crop	Isolation of *Fusarium* from 5 plants		
	16-11-1977	16-12-1977	25-1-1978
Mungbean	+ + + - -	+ - - - -	
Blackgram	- - - - -	- - - - -	
Pea	+ + - - -	+ + - - -	
French bean (*Phaseolus vulgaris*)	+ + + - -	+ + - - -	
Groundnut	+ + - - -	+ - - - -	+ - - - -
Lucern	- - - - -	- - - - -	
Lentil	- - - - -	- - - - -	
Soybean	- - - - -	- - - - -	
Cowpea	+ + + - -	+ + - - -	+ + - - -
Pigeonpea (ICP-6997)	+ + + + -	+ + + + +	+ + + + -
Pigeonpea (NP(WR)-15)	+ + + - -	+ + + - -	+ + + - -
Sorghum (CHS-1)	- - - - -	- - - - -	- - - - -
Climbing bean (*Dolichos lablab*)	- - - - -	- - - - -	- - - - -
Chilli			
Tomato			
Pearl millet (NHB-3)			
Pearl millet (HB-3)			

+ Isolated
- Not isolated

Table 9. Detection of *Fusarium* in the roots of several weed species found growing naturally in the chickpea wilt-sick plots

Weed	Isolation of *Fusarium*[a]				
	7-11-'77	11-11-'77	18-11-'77	28-12-'77	16-1-'78
Amaranthus virdis	+	-	-	x	x
Hibiscus parduraeformis	+	-	-	x	x
Phyllanthus niruri	+	-	-	-	-
P. medenaspatensis	-	-	-	x	x
Corchorus olitorius	-	-	-	-	-
Digera arvensis	-	-	-	-	-
Launea asplenifolia	-	-	-	x	x
Xanthium strumarium	-	-	-	-	x
Cyanotis axillaris	-	-	x	x	x
Euphorbia prostata	-	-	-	-	-
E. hirta	-	-	-	-	-
Indigofera sp.	-	-	-	x	x
Convolvulus sp.	-	-	-	x	x
Cassia sp.	-	-	-	x	x
Cyperus rotundus	+	+	-	+	+
Commelina bengalensis	-	-	-	x	x
Paspalum distichum	-	-	x	x	x
Eragrostis sp.	-	-	x	x	x
Desmodium triflorum	-	-	-	x	-
Heliotropium sp.	-	-	-	x	-
Tribulus terestris	+	-	+	+	x
Cardiospermum halicacabrum	-	-	+	+	x
Convolvulus arvensis	-	-	+	+	+
Lucas aspera	x	x	x	-	-
Argemone mexicana	x	x	x	-	-

+ : Present
- : Absent
x : Not attempted
[a]Five plants were used. Even if a single plant yield *Fusarium*, + sign has been indicated.

Fusarium isolates, isolated from crop plants grown in the wilt-sick plot as well as from weeds, were multiplied in the laboratory on potato-sucrose broth and tested for pathogenicity using 'water culture' technique and the susceptible JG-62 cultivar of chickpea. Although the results with regard to certain plant species tallied with those obtained through laboratory tests, the *Fusarium* (Fusaria) from field grown plants proved non-pathogenic. This is intriguing and will be investigated further.

9. Screening techniques

(i) Water culture

The "water culture" technique is similar to the procedures described by Wensley and McKeen (1962) and Roberts and Kraft (1971). The steps are:

1. An isolate of *Fusarium oxysporum* f. sp. *ciceri*, most predominant in ICRISAT fields, is used for inoculations. The culture was single spored originally and is being maintained.
2. Inoculum is multiplied on PD broth (100 ml) in flasks (250 ml) on a shaker for 10 days at room temperature (25^{o}-30^{o}C).
3. The inoculum (entire contents of the flask) is diluted with sterilized distilled water to get a final inoculum concentration of 2.5% (spore concentration - 6.5×10^{5}).
4. Seedlings 14 to 18 days old, raised in autoclaved sand, are transferred to glass tubes containing 20 ml of

inoculum. Seedlings are held in position by cotton plugs. Sterilized distilled water is filled in tubes after every 48 hr to make up the loss of water.

5. Ten seedlings are used for each line/cultivar. A susceptible check cultivar (JG-62) is likewise inoculated with each batch of test lines. Also for each line/cultivar, a noninoculated seedling is kept as check.

6. The susceptible check usually wilts between 7-10 days. Data are recorded 15 days after inoculations. Non-inoculated seedlings remain green for more than three weeks.

(ii) Pot screening

The procedure we have followed is similar to that described under pigeonpea wilt. The only difference is that we use vertisol instead of alfisol. After incorporation of inoculum, susceptible cv. JG-62 is grown and wilted plants are incorporated in the soil of these pots. Once more the same procedure is followed. After two such cycles, the pots are ready for use in screening. This procedure, like the water culture technique, is being used to supplement field screening and in assisting breeders in inheritance studies.

(iii) Sick plot

In contrast to pigeonpea wilt-sick plots, we had an easy time in developing wilt-sick plots in case of chickpea. In 1975-76 season, wilt appeared in a corner of a 2 ha block. By the end of

C CKP SAR OXYSPOR SCREE G A CR SA

1976-77 season, the whole plot developed into almost a uniform sick plot. We incorporated all the dead plants in the same plot and had excellent screening in 1977-78. We made use of this information and have developed sick plots totalling about 4 ha by growing susceptible cultivars and incorporating dead plants.

One particular plot of about 1.0 ha has been developed as a multiple disease sick plot. In this plot we have been adding every year all dead plants of that plot as well as from other plots, regardless of the cause of death. We have been having substantial infection in this plot by *Sclerotium rolfsii, Rhizoctonia solani, Rhizoctonia bataticola,* etc. in addition to *F. oxysporum* f. sp. *ciceri*. The last one is the most common fungus in our fields at ICRISAT Center.

In our wilt-sick plots, we cannot exclude the build up of other soil-borne pathogens and therefore we expect as time passes, all our plots will be multiple disease sick plots.

10. Screening work

Table 10 summarizes our work.

Table 10. Screening of chickpea for wilt/root rots resistance

Germplasm screened in wilt-sick plot	6,000
Promising against wilt	120
Germplasm screened in multiple disease nursery	1,300
Promising against wilt/root rots	80
Lines being tested in International Chickpea Root Rots/Wilt Nursery (19 countries/37 locations)	63
Breeding materials	
Screened	3,300
Promising	175

11. Resistant lines

We consider the following lines/cultivars to be resistant to Fusarium wilt:

ICC-202, ICC-391, ICC-658, ICC-858, ICC-1443, ICC-1450, ICC-1611, ICC-3439, ICC-4552, NEC-790, WR-315, CPS-1, JG-74, and BG-212

Work on wilt resistance has been done mainly at Kanpur (Singh et al. 1974) and at Jabalpur (Sharma and Khare, 1969). The sick plot screening at Gurdaspur is mainly against *Operculella padwickii*, the foot rot organism (Singh and Bedi, 1974). Incidentally WR-315 referred to above is a resistant line from Kanpur. Some work has been done in Mexico (Lopez Garcia, 1974).

12. Existence of physiologic races

Chauhan (1962) seems to be the only worker who made attempts to study variation in this pathogen. He studied 22 isolates and grouped them into five groups on the basis of filtrate toxicity and percent mortality in pot inoculations. He, however, did not specify them as races.

Preliminary studies have provided us evidence of the existence of races. The pot culture procedure was followed to study the pathogenicity of five isolates of *F. oxysporum* f. sp. *ciceri* collected from as many locations (Hyderabad, Hissar, Jabalpur, Kanpur, Gurdaspur). Ten genotypes, 4 resistant and 6 susceptible to the Hyderabad (ICRISAT) isolate, were used. The test was conducted three times and reactions in most cases were consistent. Summarized results have been presented in Table 11.

Table 11. Reactions of chickpea cultivars to five isolates of *Fusarium oxysporum* f. sp. *ciceri*[a,b]

Cultivars \ Isolates from	ICRISAT Hyderabad	Hissar	Jabalpur	Kanpur	Gurdaspur
JG-62	S	S	S	S	M
C-104	S	S	S	S	R
BG-212	R	M	M	S	M
JG-74	R	R	R	S[c]	R
CPS-1	R	M	M[d]	S	S
WR-315	R	R	R	R	S[c]
Annigeri	S	S	S	S	S
Chafa	S	S	S	S	M[d]
L-550	S	S	S	S	M
850-3/27	S	M	M	M	M

[a] 20 seedlings were used in each test and test was carried out 3 times.
[b] R = Resistant (less than 20% wilt)
M = Moderately susceptible (20-50% wilt)
S = Susceptible (more than 51% wilt)
[c] Showed 'S' reaction in two tests and 'M' in one.
[d] Showed 'M' reaction in two tests and 'S' in one.

A critical look at the results in Table 11 reveals that C-104 is resistant to the Gurdaspur isolate but susceptible to all others. JG-74 is resistant to all isolates except the Kanpur isolate. CPS-1 is resistant only to the ICRISAT isolate. WR-315 is resistant to all isolates except the Gurdaspur isolate. JG-62, Chafa, and L-550 are susceptible to all isolates and moderately susceptible to Gurdaspur

isolate. 850-3/27 is susceptible to the ICRISAT isolate and moderately susceptible to all others.

The Gurdaspur isolate was differentiated from others through resistance of C-104 and susceptibility of WR-315. The Kanpur isolate was differentiated through susceptibility of JG-74. If 'R' and 'M' categories are considered as not too distinct, the ICRISAT, Hissar, and Jabalpur isolates could be considered identical; on the other hand, if these categories are considered distinct, then the Hissar and Jabalpur isolates only could be considered identical and the ICRISAT isolate a distinct one. The data indicate that we may have 3 or 4 distinct races.

However, before we draw conclusions on this aspect, we would like to verify how serious these isolates are in field conditions at respective locations. Kraft and Haglund (1978) have emphasized this aspect in their paper on *F. oxysporum* f. sp. *pisi*.

III. Other Pathogens

Most of the literature on other soil-borne fungi deals with disease identification and prevalence. Almost no work has been done on the epidemiology of these organisms in relation to the diseases they cause in chickpea and on host resistance.

We have learned from surveys in chickpea growing countries that Ascochyta blight and stunt are widely prevalent, but these do not fall within the scope of our present review. As far as the soil-borne diseases are concerned, after wilt, dry root rot caused by *Rhizoctonia bataticola* is a relatively major problem, particularly where day time

temperatures rise to 30°C in the post-flowering stage. All other fungi discussed below are generally present, but are more of local importance, the incidence varying from field to field.

In general we observe more diseases at experiment stations than in farmers' fields. This we attribute to certain factors in farmers' fields such as rotations, mixed cropping pattern, and wide spacings because of broadcast sowings. Once high yielding cultivars are available to farmers, many of the above things will change. There will be more monocropping of chickpea, which might mean more soil-borne diseases unless resistant cultivars are made available right from the beginning. Our efforts to identify good lines under multiple disease and multi-location testing situations represent a step in that direction. For location specific diseases, the germplasm collection of ICRISAT will be made available to concerned pathologists for identifying resistance.

In the following paragraphs we have discussed other soil-borne fungi. Symptoms have been mentioned earlier.

1. *Rhizoctonia bataticola* (Dry root rot)

The pathogen does cause substantial mortality and loss in a crop which gets caught in higher ambient temperatures (30°C and above) in the post-flowering stage. In the Indian situation, this occurs in central and southern India and we see more dry root rot. It is insignificant in northern India where cooler temperatures extend through March and by the time temperatures rise, the crop is ready for harvest.

We have been making attempts to develop a laboratory screening procedure based on root lesion length as the criterion for comparing genotypes. We are hopeful that we will be able to standardize a procedure in the near future.

Dry root rot in ICRISAT Center sick plots is common in the post-flowering stage. Our screening does help us in identifying highly susceptible cultivars.

We find that alfisol extract medium supports less sclerotia production than vertisol extract medium. The dry root rot is observed more in vertisol at ICRISAT Center in both pigeonpea and chickpea.

We have observed low incidence of this disease in Lebanon, Syria, Turkey, and Iran.

2. *Rhizoctonia solani* (Root rot)

It has never been reported to be serious from any chickpea growing area. Most of the incidence is seen in the seedling stage when soil moisture content is high. In irrigated chickpeas, the disease may occur any time. We have seen this disease more frequently in chickpeas planted after the harvest of paddy where soil moisture content is higher.

We have seen this disease occasionally in our multiple disease nursery at ICRISAT Center.

3. *Sclerotium rolfsii* (Collar rot)

The incidence is related to higher moisture content and presence of undecomposed organic matter near soil surface. It is a problem in the seedling stage except in irrigated crops where the disease can

occur at any stage provided temperatures are not low. Chickpea following paddy shows more incidence.

Our multiple disease sick plot shows some incidence of collar rot every year. At Jabalpur, where the crop in the sick plot is irrigated, the collar rot incidence is relatively higher.

4. *Sclerotinia sclerotiorum* (Stem rot)

The problem is seen in northern India where cool temperatures, relatively more rain in January, and heavy dew occur which are favourable to the pathogen. The disease does cause substantial damage if plantings are close and the crop canopy is thick. In case of more rains in a season, the vegetative growth of chickpea becomes excessive. In such years this disease can become serious.

No attempt to identify resistance to this disease has been made.

In addition to India, the disease has been reported from Chile (Mujica, 1955) and Iran (Kaiser, 1972).

5. *Operculella padwickii* (Foot rot)

Kheswalla (1941) described this disease first from Punjab and Delhi in northern India. Although the fungus has been isolated from several locations in central and northern India, the disease seems to be location specific. At Gurdaspur in northern India, this fungus is the most dominant one in the sick plot. We feel wet soil is conducive to this disease. From Gurdaspur, Singh and Bedi (1974) reported that G-543 is a resistant cultivar and F-61 is moderately resistant.

This fungus has been reported only from India.

6. *Fusarium solani* (Root rot)

Kraft (1969) first reported that *F. solani* f. sp. *phaseoli* can infect chickpea. Westerlund et al. (1974) reported it to be one of the root rotting fungi of chickpea in California. The same year Grewal et al. (1974) reported it from northern India. Although the fungus has been isolated from diseased chickpea plants from different areas of India, it is restricted mainly to northern India. The chickpea plots at New Delhi usually show more incidence of *F. solani* and screening against this pathogen should be possible there.

No specific resistance sources have yet been identified.

7. *Ozonium texanum* var. *parasiticum* (Wilt/Foot rot/Root rot?)

Mishra (1955) first reported this pathogen from Bihar state of India. He called the disease wilt although the fungus causes rotting at the base as well as of roots. So far the disease has been reported from Bihar state and the adjacent area of eastern Uttar Pradesh state.

Again there is no information on resistance to the disease.

8. A sterile fungus (white seed and root rot)

Haware and Nene (1976) have reported a sterile fungus responsible for causing seed rot as well as root rot. Thick white mycelial strands cover the seed affecting germination or cover the young roots of seedlings. The disease is observed only if the soil is too wet after sowing which happens due to chance rains.

Since the disease is a minor problem we have not done any further work.

9. *Meloidogyne* spp. (Root-knot)

The problem has been seen mainly in irrigated chickpeas. More incidence has been noted in northern India. A good root-knot infested plot at Ludhiana offers an excellent opportunity to screen for resistance. After the problem was identified at Ludhiana, there has been increased interest in this problem amongst the nematologists in northern India.

One of the species identified is *M. incognita* (Ahmad Jamal, 1976).

LITERATURE CITED

Agnihotrudu, V. 1954. Soil conditions and wilt diseases in plants. Rhizosphere microflora in relation to fungal wilts. Ph.D. thesis submitted to Madras Univ. (cf. Sadasivan T.S., and C.V. Subramanian, 1954. Recent advances in the study of soil-borne Fusaria. J. Indian Bot. Soc. 33: 162-176).

Ahmad Jamal. 1976. Studies on the relationship between *Meloidogyne incognita* and behaviour of *Cicer arietinum* roots. Curr. Sci. 45: 230-231.

Amin, K.S., B. Baldev, and F.J. Williams. 1978. *Phytophthora cajani*, a new species causing stem blight on *Cajanus cajan*. Mycologia (under print according to the senior author).

Anonymous. 1953. Gram wilt and its control. Indian Council Agr. Res., New Delhi and Indian Fmg. 3: 7.

Armstrong, G.M., and Joanne K. Armstrong. 1948. Nonsusceptible hosts of carriers of wilt Fusaria. Phytopathology 38: 808-826.

Baldev, B., and K.S. Amin. 1974. Studies on the existence of races in *Fusarium udum* causing wilt of *Cajanus cajan*. SABRAO J. 6: 201-205.

Bedi, K.S., and C.S. Pracer. 1952. Gram wilt disease and its control in Punjab. Punjab Farmer 4: 296-301.

Booth, C. 1971. The genus *Fusarium*. Commonwealth Mycological Institute, Kew, (Surrey) England. pp.1-137.

Bose, R.D. 1938. The rotation of tobacco for the prevention of wilt disease in pigeonpeas (*Cajanus cajan*(Linn.) Millsp.). Agr. Livestock, India 8: 6 : 653-668.

Butler, E.J. 1906. The wilt disease of pigeonpea and pepper. Agr. J. India 1: 25-36.

Butler, E.J. 1908. Selection of pigeonpea for wilt disease. Agr. J. India 3: 182-183.

Butler, E.J. 1910. The wilt disease of pigeonpea and the parasitism of *Neocosmospora vasinfecta* Smith. Mem. Dep. Agr. India, Bot. Ser. 2: 1-64.

Butler, E.J. 1918. Fungi and plant disease. pp. 1-547. Reprinted in 1973 by M/s. Bishen Singh Mahendra Pal Singh, Dehradun and M/s. Periodical Experts, Delhi.

Chattopadhyay, S.B., and P.K. Sen Gupta. 1967. Studies on wilt diseases of pulses. I. Variation and taxonomy of *Fusarium* species associated with wilt disease of pulses. Indian J. Mycol. Res. 5: 45-53.

Chauhan, S.K. 1960. A technique for disease appraisal and loss estimations in Fusarium wilt of gram. Agra Univ. J. Res. 9: 253-260.

Chauhan, S.K. 1962. Physiologic variations in *Fusarium orthoceras* App. & Wr. var. *ciceri* Padwick causing wilt of gram (*Cicer arietinum* L.). Proc. Nat. Acad. Sci. India. Sect B. 32: 78-84.

Dastur, J.F. 1935. Gram wilts in the Central Provinces. Agr. Livestk. India 5: 615-627.

Deshpande, R.B., L.M. Jeswani, and A.B. Joshi. 1963. Breeding wilt resistant varieties of pigeonpea. Indian J. Genet. 23: 58-63.

Dey, P.K. 1948. Plant Pathology. Administrative Rep. Agr. Dep. U.P., 1946-47. pp.39-42.

Erwin, D.C. 1958. *Fusarium lateritium* f. *ciceri*, incitant of Fusarium wilt of *Cicer arietinum*. Phytopathology 48: 498-501.

Erwin, D.C., and W.C. Snyder. 1958. Yellowing of Garbanzo Beans. Calif. Agr. 12: 6: 16.

Grewal, J.S., M. Pal, and D.D. Kulshrestha. 1974. Fungi associated with gram wilt. Indian J. Genet. & Plant Breeding 34: 242-246.

Grewal, J.S., M. Pal, and D.D. Kulshrestha. 1974. A new record of wilt of gram caused by *Fusarium solani*. Curr. Sci. 43: 767.

Haware, M.P., and Y.L. Nene. 1976. Some uncommon but potentially serious diseases of chickpea. Tropical Grain Legume Bull. No.5: 26-30.

Jain, H.K., and P.N. Bahl. 1974. Recommendations of symposium on gram wilt. Indian J. Genet. & Plant Breeding 34: 236-238.

Kaiser, W.J. 1972. Occurrence of three fungal diseases of chickpea in Iran. FAO Plant Prot. Bull.20: 74-78.

Kaiser, W.J., and D. Danesh. 1971. Etiology of virus-induced wilt of Cicer arietinum. Phytopathology 61: 453-457.

Kheswalla, K.F. 1941. Foot rot of gram (*Cicer arietimum* L.) caused by *Operculella padwickii* nov. gen. nov. sp. Indian J. Agr. Sci. 11: 316-318.

Kraft, J.M. 1969. Chickpea, a new host of *Fusarium solani* f. sp. *pisi*. Plant Dis. Reptr. 53: 110-111.

Kraft, J.M., and W.A. Haglund. 1978. A reappraisal of the race classification of *Fusarium oxysporum* f. sp.*pisi*. Phytopathology 68: 273-275.

Lopez Garcia, H. 1974. Inheritance of the character resistance to wilt (*Fusarium* sp.) in chickpea (*Cicer arietinum*) under field conditions (in Spanish). Agricultura Tecnica Mex. 3: 286-289.

McKerral, A. 1923. A note on Fusarium wilt of gram in Burma and measures taken to combat it. Agr. J. India 28: 608-613.

McRae, A.T.K., and F.J.F. Shaw. 1933. Part I. Influence of superphosphate, cattle manure and green manure on the wilt of *Cajanus indicus* caused by *Fusarium vasinfectum* Atk. Part II. The isolation of resistant types. Imperial Coun. Agr. Res. Scientific Monograph, Vol.7: pp.1-68.

Miller-Jones, D.N., D.R. Houston, and T.F. Preece. 1977. The use of electrical resistance measurements to detect watermark disease of cricket bat willow. Plant Dis. Reptr. 61: 268-272.

Mishra, J.N. 1955. Wilt of gram (*Cicer arietinum* L.) in Bihar. Curr. Sci. 24: 210.

Mohanty, U.N. 1949. The wilt disease of pigeonpea with special reference to the distribution of causal organism in the host tissue. Indian J. Agr. Sci. 16: 379-390.

Mujica, R.F. 1955. Studies on sclerotiniosis (in Spanish). Agricultura Tec., Santiago 15: 64-74.

Narasimhan, R. 1929. A preliminary note on a Fusarium parasitic on Bengal gram (*Cicer arietinum*). Madras Agr. Dep. Year Book 1928: 5-11.

Padwick, G.W. 1940. The genus Fusarium. III. A critical study of the fungus causing wilt of gram (*Cicer arietinum* L.) and of the related species of the subsection *Orthoceras*, with special relation to the variability of key characteristics. Indian J. Agr. Sci. 10: 241-284.

Pal, Mahendra, J.S. Grewal, and A.K. Sarbhoy. 1970. A new stem rot of arhar caused by *Phytophthora*. Indian Phytopath.23: 583-587.

Prasad, N., and G.W. Padwick. 1939. The genus Fusarium. II. A species of *Fusarium* as a cause of wilt of gram (*Cicer arietinum* L.). Indian J. Agr. Sci. 9: 371-380.

Raut, N.K., and B.B. Bhombe. 1971. A review of the work of selections of tur varieties resistant to Fusarium wilt at College of Agriculture, Parbhani (Maharashtra). Magazine College of Agr. Parbhani 12: 37-42.

Roberts, D.D., and J.M. Kraft. 1971. A rapid technique for studying Fusarium wilt of peas. Phytopathology 61: 342-343.

Sarojini, T.S. 1950. Soil conditions and root diseases. Part I. Micro-nutrient elements and disease development by *Fusarium udum* on red gram (*Cajanus cajan* (Linn.) Millsp.). J. Madras Univ. B. 19: 1-32.

Sarojini, T.S. 1951. Soil conditions and root diseases. Part II. *Fusarium udum* disease of red gram (*Cajanus cajan* (Linn.) Millsp.). Proc. Indian Acad. Sci. Section B, 33: 49-68.

Sharma, H.C., and M.N. Khare. 1969. Studies on wilt of Bengal gram (*Cicer arietinum* L.) at Jabalpur. JNKVV Res. J. 3: 122-123.

Sharma, R.D., and R.S. Singh. 1973. A technique for selective isolation of *Fusarium moniliforme* from soil and plant tissues. Indian J. Mycol. & Plant Path. 3: 67-70.

Shukla, D.S. 1975. Incidence of Fusarium wilt of pigeonpea in relation to soil composition. Indian Phytopath. 28: 395-396.

Singh, D.V., A.N. Misra, and S.N. Singh. 1974. Sources of resistance to gram wilt and breeding for wilt resistance in Bengal gram in U.P. Indian J. Genet. & Plant Breeding 34: 239-241.

Singh, G., and P.S. Bedi. 1975. The perpetuation of *Operculella padwickii*, the cause of foot blight (rot) of gram in the Punjab. Indian Phytopath. 28: 546-548.

Singh, K.B., and B.S. Dahiya. 1973. Breeding for wilt resistance in chickpea. Symposium on wilt problem and breeding for wilt resistance in Bengal gram. September 1973 at Indian Agr. Res. Inst., New Delhi, India, pp.13-14 (Abstr.).

Snyder, W.C., and H.N. Hanson. 1940. The species concept in Fusarium. Amer. J. Bot. 27: 64-67.

Subramanian, C.V. 1955. Studies on south Indian Fusaria. IV. The "wild type" in *Fusarium udum* Butler. J. Indian Bot. Soc. 34: 29-36.

Vaheeduddin, S. 1958. Evolving wilt resistant strains of red gram. Andhra Agr. J. 5: 163-164.

Wensley, R.N., and C.D. McKeen. 1962. Rapid test for pathogenicity of soil isolates of *Fusarium oxysporum* f. *melonis*. Can. J. Microbiol. 8: 818-819.

Westerlund, F.V. Jr., R.N. Campbell, and K.A. Kimble. 1974. Fungal root rots and wilt of chickpea in California. Phytopathology 64: 432-436.

Williams, F.J., J.S. Grewal, and K.S. Amin. 1968. Serious and new diseases of pulse crops in India in 1966. Plant Dis. Reptr. 52: 300-304.

Williams, F.J., K.S. Amin, and B. Baldev. 1975. Phytophthora stem blight of *Cajanus cajan*. Phytopathology 65: 1029-1030.

APPENDIX-I

Development of pigeonpea wilt-sick plots

Vertisol sick plot 'A' (1.5 ha)

March 11, 1975	:	Added 750 cu.ft. of compost (chopped stubble) of field-wilted pigeonpea, pod husk and sorghum heads after these were composted together for 1 or 2 months.
May 1st week, 1975	:	Again added 750 cu.ft. of compost as described above.
June 26, 1975	:	Incorporated 1.87 q of sorghum grain colonized by pigeonpea *Fusarium*.
April 30, 1976	:	Scattered 7.50 q of *Fusarium* colonized pigeonpea seeds.
May 5, 1976	:	All the wilted plants stubble were chopped and incorporated into soil.
July 12, 1976	:	Incorporated 11.25 q of *Fusarium* multiplied on alfisol + pigeonpea flour (9:1 w/w).
April 5, 1977	:	All the wilted plants stubble were chopped and incorporated into soil.
May 2, 1978	:	All the wilted plants stubble were chopped and incorporated into soil.
May 8, 1978	:	Scattered uniformly about 500 cu.ft. of wilted pigeonpea stem bits.

Vertisol sick plot 'B' (1.5 ha)

April 19&20, 1976	:	Scattered 1,500 cu.ft. pigeonpea stem bits (both wilted and healthy plants).
April 30, 1976	:	Scattered 7.50 q of *Fusarium* colonized pigeonpea seeds.
July 12, 1976	:	Incorporated 11.25 q of *Fusarium* multiplied on alfisol + pigeonpea flour (9:1 w/w).
July 29, 1976	:	Pigeonpea wilted stem pieces (15 cm) were buried in every row (one piece after every two plants).
February & March, 1977	:	Scattered 400 cu.ft. wilted pigeonpea stem bits.
April 6 & 7, 1977	:	All the wilted plants stubble of 1976-77 were chopped and incorporated into soil.
May, 1978	:	All the wilted plants stubble of 1977-78 were chopped and incorporated into soil.
May, 1978	:	Scattered uniformly about 500 cu.ft. of wilted pigeonpea stem bits.

Alfisol sick plot 'A' (0.1 ha)

- This plot was used as pigeonpea sterility mosaic screening nursery for three years (1974-77) continuously. During that period increased wilt incidence was observed every year.

- In 1977-78 the plot was used to screen pigeonpea for wilt and sterility mosaic diseases. Wilt susceptible check line (ICP-6997) showed 99.4 percent disease.
- All wilted plants stubble of 1977-78 were chopped and incorporated into soil (April 24, 1978).

Alfisol sick plot 'B' (0.4 ha)

1977-78	:	Planted pigeonpea materials for sterility mosaic screening.
January, 1978	:	Wilt incidence was observed in large patches.
April, 1978	:	All the wilted plants were chopped and incorporated into soil.
May, 1978	:	Scattered about 400 cu.ft. of pigeonpea wilted stem bits.

YLN:prm
2211'78

CONSULTANTS' GROUP DISCUSSION ON

THE RESISTANCE TO SOIL-BORNE DISEASES OF LEGUMES

(January 8-11, 1979)

SOME POINTS FOR DISCUSSION

Pigeonpea wilt

1. Have our studies on the survival of *Fusarium udum* in pigeonpea stubble been carried out adequately? (Please see pp.8-10)
2. What could be the reasons for the failure of water culture screening technique in case of pigenpea but not in chickpea? (pp.11 & 42)
3. The technique of transplanting seedlings, roots of which are injured and inoculated, to autoclaved sand/soil in pots gave us erratic results. What could be the reasons? (p.11)
4. We would appreciate comments/criticism on the pot screening procedure developed by us. (pp.11-12)
5. We have developed two wilt-sick plots in vertisol for resistance screening (pp.12-13):
   (a) Is it possible that the plots may contain 'too much' inoculum as the years pass by?
   (b) Are we likely to face other problems?
   (c) We are using mainly one susceptible check (ICP-6997) to monitor wilt sickness. Is that adequate?

(d) The susceptible check rows are planted after every 2 to 4 test rows also to ensure that inoculum multiplies every year. Is this adequate or should we follow the procedure of growing only a susceptible cultivar one year and test material in the next year (with a few check lines)? The two sick plots that we have developed can be used in such a way that when one has only the susceptible cultivar, the other would have the breeding material.

(e) There are indications that continuous planting of pigeonpea is resulting in poorer growth in every succeeding season. This is likely to result into rejection of breeding material which may be resistant but showing poor growth in sick plot. What could be done to avoid such a situation?

6. Our experience tells us that wilt sickness can be developed more quickly and uniformly in alfisol than in vertisol. We have developed two large sick plots in vertisol because farmers prefer this type of soil (i.e. deep soils) for cultivating pigeonpea. We find that some genotypes which show 'resistance' in vertisol get affected by wilt in alfisol, but the reverse has never happened. Should we therefore develop sick plots in alfisol and give up the existing sick plots in vertisol? Or should we have large sick plots in both types of soil? We must mention here that

to grow pigeonpea irrigation is required in alfisol but not in vertisol.

7. We consider multilocation testing of promising lines desirable before using them in crosses. Is our thinking correct?
8. Since the wilt incidence increases considerably after ratooning, is it desirable to go by the post-ratoon reaction of lines? (p.15)
9. What are the possibilities of developing a selective medium for *Fusarium udum?*

Pigeonpea Phytophthora blight

1. Our observations concerning the survival of the fungus have been described on pages 18 & 22. We need suggestions to plan research on this aspect.
2. We would appreciate comments/criticism on the pot screening procedure we have developed (pp.22-23).
3. We need suggestions to improve upon our field screening procedure (pp.23-24).

Chickpea wilt/root rots

1. Many plant species grown in the wilt-sick plot yielded *Fusarium*, which morphologically looked similar to the isolate of *F. oxysporum* f. sp. *ciceri*. However, *Fusarium* isolates from all these plant species were non-pathogenic to chickpea. We will appreciate discussion on this point (pp.39-42).
2. We will appreciate comments/criticism on water culture and pot culture screening techniques for wilt resistance (pp.42-43)

3. Several soil-borne pathogens which can attack chickpea are present in most soils, even though one or two pathogens may dominate. In sick plots at ICRISAT *Fusarium oxysporum* f. sp. *ciceri* dominates, but other pathogens such as *Rhizoctonia bataticola* also kill many lines (pp.43-44). Should we therefore encourage 'multiple disease sick plots' and identify lines which show least mortality for use in the breeding program? Or should we concentrate on working out procedures for identifying resistances to different soil-borne pathogens individually?
4. Pathogens other than *Fusarium oxysporum* f. sp. *ciceri* are important at other locations. For example *Operculella padwickii* is the dominant fungus at Gurdaspur. How should we conduct work to meet such situations?
5. Evidence indicates that physiologic races of *Fusarium oxysporum* f. sp. *ciceri* exist (pp.45-47). Is multilocation testing of our promising lines the only answer to meet this situation?
6. Dry root rot caused by *Rhizoctonia bataticola* is another widely prevalent disease. We are making attempts to develop a laboratory screening procedure based on root lesion length. We invite your comments/criticism/suggestions? (pp.48-49)
7. We may have to work out techniques to screen for resistance to root rots caused by *Fusarium solani* and *Rhizoctonia solani*. We would appreciate suggestions.